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Lead in Soil: Recommended Maximum Permissible Levels¹

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INTRODUCTION

Environmental exposure to lead has long been recognized as a public health problem particularly among children. The vulnerability of the age group 1 to 5 years to soil lead is enhanced because of their hand to mouth activities, pica, and a high rate of intestinal absorption. Excessive concentration of lead in soil has been shown to increase lead levels in children (Lin-Fu, 1973a, b; Mielke *et al.*, 1983; Duggan and Williams, 1977; Brunekreef *et al.*, 1981; Roels *et al.*, 1980; Schmitt *et al.*, 1979). As a result, there has been an increasing awareness for the need to monitor lead levels in soil and to control soil lead contamination by maintaining a "safe" level. Given the widespread presence of lead in urban soil, reduction of lead to background uncontaminated levels is not possible (National Academy of Sciences, 1980). The major focus of this report is to propose a "safe" or permissible level of lead in soil in highly urbanized areas, below which potential adverse health effects will be minimized.

BACKGROUND

Environmental Assessment

Soil lead contamination has been attributed to various sources (American Academy of Pediatrics, 1985). Flaking lead paint, particularly in and around houses or buildings has been considered as a major source of contamination. Air-borne lead particles deposited in soil is another important source. Emissions from industries, from incinerators and similar sources, and from vehicular traffic using leaded gasoline contribute to soil lead content. Urban environments receive a higher deposition of lead from vehicular emissions than rural areas. Furthermore, lead concentration in urban soils are not evenly distributed (Mielke *et al.*, 1983).

In general, lead tends to remain at the surface soil and concentrations are lower at deeper layers. Lead-contaminated soil and dust have been identified as important sources of exposure for children especially in an urban setting (Duggan and Williams, 1977). Wide variations in soil lead levels have been observed. Studies

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have reported values ranging from less than 100 ppm to well over 11,000 ppm (National Academy of Sciences, 1980). In a recent study in Baltimore, the lead levels in garden soil samples ranged from 1.0 ppm to over 10,000 ppm with a median of 100 ppm (Mielke *et al.*, 1983). Spittler and his co-workers did a similar study on garden soil in Boston (Spittler and Feder, 1979). Soil lead levels were higher in inner-cities and near roadways. Also, front yards of homes facing roadways had higher lead contamination than backyards. Automobile and industrial emissions have been found to be mainly responsible for increase in urban soil lead levels.

Health Effects

Severe lead toxicity often causes encephalopathy. Prevention of this serious sequelae of lead poisoning was a major focus in the 1960s (Mahaffey, 1983). During the 1970s, recognition of chronic exposure of lead and its cumulative effect shifted the emphasis to the understanding of the adverse effects of low levels of lead intoxication. The study by Needleman *et al.* (1979) showed a positive relationship of lead in shed milk teeth with poor ratings from classroom behavior. These findings supported the "no threshold" view and also indicated the need for more attention to be given to cumulative adverse effects of lead at low levels of intake. A recent study in Boston (Bellinger *et al.*, 1987) emphasized this view with its findings on fetal lead exposure associated with retardation of mental development.

The blood lead concentration has been generally accepted as the best measure of the external dose of lead (National Academy of Sciences, 1980), although it is not considered as a reliable index of past absorption or of toxicity per se. However, Needleman *et al.* (1979) had observed that children with higher tooth lead levels tended to have had higher blood lead levels previously (4 or 5 years prior to tooth shedding).

In recent years, progress has been made in achieving the goal to remove lead from the environment of children before it enters their bodies. The Second National Health and Nutrition Examination Survey (NHANES-II) has established average blood lead levels for the U.S. population (Mahaffey *et al.*, 1982). These data demonstrated that urbanization was associated with an increased blood level. Lead levels in blacks were on an average 6 µg/dl higher than those in whites. The lowest blood lead associated with adverse biological effects has been observed to be 10 µg/dl (Minnesota Department of Health, 1984). ALAD (Δ-aminolevulinic acid dehydratase) inhibition is associated with this low level. More serious conditions such as anemia and neurologic effects occur at higher levels of blood lead elevation.

Leaded gasoline makes a substantial contribution to soil and dust lead levels (Caprio *et al.*, 1974). The reduction of lead in gasoline and removal of lead in paint for residential areas have been primarily responsible for a decline in the average blood lead levels in children on a national basis. In areas with very high concentrations of lead in soil and dust, large-scale cleanup operations of the population will be the ideal remedial actions to protect



lead exposure. Such responsibilities for regulating lead exposure include the setting up of acceptable levels of lead in soil by government agencies.

MATERIALS AND METHODS

Several studies have found that lead in soil is positively correlated with blood lead in children (Brunekreef, 1981; Roels *et al.*, 1980; Schmitt *et al.*, 1979). The U.S. Environmental Protection Agency (EPA) estimated the blood lead soil slope as ranging from 0.6 to 6.8 $\mu\text{g}/\text{dl}$ per 1000 $\mu\text{g}/\text{g}$ of soil lead concentration (U.S. Environmental Protection Agency, 1983). Available data on the estimates of the amount of soil ingested by children showed 100-fold variation and were not considered useful in deriving a "safe" soil level (Binder *et al.*, 1986; Clausen *et al.*, 1987; Hawley, 1985).

Duggan (1980) did an assessment of the relationship of blood lead and lead in soil/dust, based on 21 samples out of nine studies, which had data permitting a quantitative estimation of the blood lead slope. His estimate was an increment of 5 $\mu\text{g}/\text{dl}$ of blood lead per 1000 ppm of lead in soil. These studies varied a great deal in the type of soil and the study population. Soil or dust source included various types such as boot tray dust, house dust, outdoor dust, playground dust, and soil. Most of these studies were on children under 5 years of age, a few on older children up to 14 years, and one on a mixed population of adults and children. The blood lead slopes, computed by Duggan for all 21 samples, were available, ranging from 0.6 $\mu\text{g}/\text{dl}$ to 65 $\mu\text{g}/\text{dl}$ per 1000 ppm of lead in soil.

We based our analysis on 8 of Duggan's 21 slope estimates. We selected these 8 slopes because soil was the only source of lead, not house dust, etc., and only blood levels from children under 12 years, the most susceptible group to lead toxicity, were used to derive the slopes.

RESULTS AND DISCUSSION

The results of limited soil sampling in New Jersey found that median values of lead in surface soil samples from different areas in New Jersey varied from 4 ppm to 1245 ppm (New Jersey State Department of Health, 1985). The overall median levels were 238 and 73 ppm for suspected contaminated and control sites, respectively. Newark had the highest median of 1245 ppm followed by Jersey City (668 ppm), Secaucus (495 ppm), and other towns with levels below 400 ppm. Samples from areas in Princeton and Flemington were below 100 ppm. As observed in earlier studies, front yards of homes in Newark had a higher level (1755 ppm) than backyards (1060 ppm).

Table 1 shows the slopes ranging from 0.6 to 65 $\mu\text{g}/\text{dl}$ per 1000 ppm of the eight studies selected to derive an acceptable level for lead in soil. As lead levels in blood are known to be distributed lognormally, and the range for slopes (0.6–65.0 $\mu\text{g}/\text{dl}$) is very wide, analysis was done on base 10 log transformations of the slopes. The mean of the base 10 logs is 0.5321 with a standard error of 0.2435. Transforming back, the geometric mean and the geometric standard error of the slopes is $3.41 \pm 1.75 \mu\text{g}/\text{dl}$. Applying the "worst-case" or upper-limit analysis to the base 10 logs, the one-tailed 95% upper confidence limit equals $0.5321 + 1.65 \times 0.2435 = 0.9339$ (American Industrial Health Council, 1985; Wilson and

TABLE 1
DATA RELATING TO LEAD IN BLOOD WITH LEAD IN SOIL*

Author and reference	Number of persons in study	Age of persons (years)	Slope ($\mu\text{g/dl}/1000 \text{ ppm}$)
Angle <i>et al.</i> (I)	153	2-5	65.0
Angle <i>et al.</i> (II)	25	10-12	15.0
Roritrop <i>et al.</i>	82	2	0.6
Galke <i>et al.</i> (i)	187	up to 5	3.3
Galke <i>et al.</i> (II)	187	up to 5	1.6
Shellehear <i>et al.</i>	68	1-5	3.9
Yankel <i>et al.</i> (i)	1149	1-9	0.6
Yankel <i>et al.</i> (II)	1149	2-3	2.5

* Source: Duggan (1980).

Crouch, 1982). Transforming back, the antilog is 0.9339-8.5877 $\mu\text{g/dl}$ per 1000 ppm of lead in soil. This slope corresponds to the worst case situation.

Using the slope 8.59 $\mu\text{g/dl}$, soil concentrations have been calculated for different amounts of blood lead contributed from soil, as shown in Table 2. Having computed the soil concentration for different amounts of blood lead contributed through soil, the next important consideration is the choice of the permissible amount of blood lead from soil. The soil lead concentration corresponding to this blood lead level would be the suggested lead permissible level. Keeping in mind the background level of blood lead for children under 12 years, the ideal situation would be to have no increment in blood lead level contributed from soil. This stringent condition demands a zero level concentration of lead in soil. Looking at estimates of soil lead levels available from various studies in the United States and elsewhere, one realizes that to bring down the lead concentration to zero would be an impractical task. As shown in Table 2, even for 1 $\mu\text{g/dl}$ of blood lead from soil, the soil concentration has to be around 100 ppm. If 5 $\mu\text{g/dl}$ of blood lead is chosen as a tolerable level, the corresponding soil concentration is 582 ppm, rounded off to a figure of 600 ppm. With a suggested permissible level of 600 ppm, it can be stated with reasonable certainty that this soil concentration will contribute no more than 5 $\mu\text{g/dl}$ to blood lead for children under 12 years. The selection of 5

TABLE 2
LEAD CONCENTRATION IN SOIL BY BLOOD LEAD CONTRIBUTION FROM SOIL

Blood lead from soil* ($\mu\text{g/dl}$)	Soil concentration (ppm) at 95% upper confidence limit of 8.59 $\mu\text{g/dl}$ 1000 ppm
1	116
5	582
10	1164
15	1746
20	2328
25	2910

* In addition to background level.

$\mu\text{g/dl}$ is somewhat arbitrary. The median blood lead of children 6 months to 5 years between 1976 and 1980 was reported to be 16 $\mu\text{g/dl}$ for whites and 20 $\mu\text{g/dl}$ for blacks. Since lead accumulates there is no absolutely "tolerable" increase of blood level. Allowing an increase of 5 $\mu\text{g/dl}$ above the median level is probably not advisable. The national median levels, however, are probably partially attributable to soil contamination. Table 2 therefore needs to be used as a guideline to the upper limit of accumulation not as a standard which if met guarantees absolute safety.

This suggested level of 600 ppm lies within the range given by the Center for Disease Control (1985) in the following statement:

"In general, lead in soil and dust appears to be responsible for blood lead levels in children increasing above background level when the concentration in the soil or dust exceeds 500-1000 ppm."

A similar analysis was done by the EPA. (U.S. EPA, 1983). In that analysis the value of 65 $\mu\text{g/dl}/1000$ ppm from one study (Angle, see Table 1) was not included. Eliminating this outlier would change the 95% upper confidence limit of the slope from 8.59 that we used, to 4.52 $\mu\text{g/dl}/1000$ ppm. This would approximately double the soil levels presented in Table 2. Eliminating the upper and lower outliers in Table 1 would not appreciably change the slope or values in Table 2. Because of the uncertainty involved in selecting a "safe" level we do not feel that it is warranted to exclude the data at either extreme.

Furthermore, it is important to keep in mind that exposure of children to lead-contaminated soil or dust is enhanced when they play on nongrassy surfaces than on grass-covered areas (Lewis and Clark County Health Department *et al.*, 1986), a scenario similar to the vulnerability of children exhibiting mouthing behavior.

In conclusion, maximum permissible levels of lead in soil have been recommended by the New Jersey State Department of Health, based on the dose-response relationship of lead in soil and blood lead in children as follows:

1. A maximum permissible level of 250 ppm of lead in soil is recommended in areas without grass cover and repeatedly used by children below 5 years of age among whom mouthing objects is highly prevalent. This level may add at the most about 2 $\mu\text{g/dl}$ to the blood lead level of children.
2. A maximum permissible level of 600 ppm of lead in soil is recommended in areas repeatedly used by children below 12 years of age. This level may add at the most 5 $\mu\text{g/dl}$ to blood lead level of children.
3. A maximum permissible level of 1000 ppm of lead in soil is recommended in areas such as industrial parks or along streets and highways or in other areas infrequented by children. Although these areas are not expected to be places where children play, we do not feel that this can always be assured. Additionally, we are concerned about migration of lead off these sites on the footwear or clothes of adults.

The Department of Health also recommends that municipalities should consider the passage of local ordinances prohibiting the development of residential areas in lead-contaminated soil unless the lead soil concentration is reduced to the appropriate maximum permissible level.

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SUMMARY

Lead in soil has been recognized as a public health problem, particularly among children. In recent years, attention has been directed to cumulative adverse effects of lead at low levels of intake. Lead-contaminated soil and dust have been identified as important contributors to blood lead levels. Based on available data on blood lead and lead in soil, an approach has been developed to suggest a permissible level of lead in soil, below which there will be reasonable certainty that adverse health effects will not occur. An acceptable level of 600 ppm of lead in soil suggested as a "safe" level would contribute no more than 5 µg/dl to total blood lead of children under 12 years of age. Maximum permissible levels of lead in soil have been recommended based on the dose-response relationship of lead in soil and blood lead in children.

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REFERENCES

- American Academy of Pediatrics. (1985) "A Statement on Childhood Lead Poisoning." Draft prepared by Committee on Environmental Hazards.
- American Industrial Health Council. (1985). "Proposals to Improve Scientific Risk Assessment for Chemical Carcinogenesis. Dose Response Evaluation and Characterization of Risk." pp. 33-38. Scientific working paper by American Industrial Health Council, Washington, DC.
- Bollinger, D., Leviton, A., Waterman, C., Needleman, H., and Rabinowitz, M. (1987). Longitudinal analysis of prenatal and postnatal lead exposure and early cognitive development. *N. Engl. J. Med.* 316, 1037-1043.
- Binder, S., Sukal, D., and Maughan, D. (1986). Estimating soil ingestion: The use of tracer elements in estimating the amount of soil ingested by young children. *Arch. Environ. Health* 41, 341-345.
- Brunekroef, B., Veenstra, S. J., Biersteker, K., and Boley, J. S. M. (1981). The Arnhem lead study. 1. Lead uptake by 1- to 3-year-old children living in the vicinity of a secondary lead smelter in Arnhem, The Netherlands. *Environ. Res.* 25, 441-448.
- Caprio, R. J., Margulis, H. L., and Joselow, M. M. (1974). Lead absorption in children and its relationship to urban traffic densities. *Arch. Environ. Health* 28, 195-197.
- Centers for Disease Control. (1985). "Preventing Lead Poisoning in Young Children." U.S. Department of Health and Human Services, Atlanta, GA.
- Clausing, P., Brunekroef, B., and van Wijnen, J. H. (1987). A method for estimating soil ingestion by children. *Int. Arch. Occup. Environ. Health* 59, 73-82.
- Duggan, M. J. (1980). "Lead in Urban Dust: An Assessment. Water, Air and Soil Pollution," pp. 309-321. Reidel, Boston.
- Duggan, M. J., and Williams, S. (1977). "Lead-in-Dust in City Streets. The Science of the Total Environment." Vol. 7, pp. 91-97. Elsevier Scientific, Amsterdam.
- Hawley, J. K. (1985). Assessment of health risk from exposure to contaminated soil. *Risk Anal.* 5, 289-302.
- Lewis and Clark County Health Department, Montana Department of Health and Environmental Sciences, Center for Environmental Health, Centers for Disease Control; Public Health Service, U.S. Department of Health and Human Services, and U.S. Environmental Protection Agency (1986). "East Helena, Montana Child Lead Study, Summer 1983. Final Report, 36."

- Lin-Pu, J. S. (1973a). Vulnerability of children to lead exposure and toxicity. Part 1. *N. Engl. J. Med.* 289, 1229-1233.
- Lin-Pu, J. S. (1973b). Vulnerability of children to lead exposure and toxicity. Part 2. *N. Engl. J. Med.* 289, 1289-1293.
- Mahaffey, K. R. (1983). Sources of lead in the urban environment. *Amer. J. Public Health* 73, 1357-1358.
- Mahaffey, K. R., Annett, J. L., Roberts, J., Murphy, R. S. (1982). National estimates of blood lead levels: United States, 1976-1980. Association with selected demographic and socioeconomic factors. *N. Engl. J. Med.* 307, 573-579.
- Mielke, H. W., Anderson, J. C., Berry, K. J., Mielke, P. W., Chaney, R. L., and Leach, M. (1983). Lead concentrations in inner-city soils as a factor in the child lead problem. *Amer. J. Public Health* 73, 1366-1369.
- Minnesota Department of Health, Division of Maternal and Child Health Services. (1984). "Lead Exposure and the Health Effects on Children." Report to the Minnesota Legislature.
- National Academy of Sciences. (1980). "Lead in the Human Environment." Report prepared by the Committee on Lead in the Human Environment, National Research Council. Natl. Acad. Press, Washington, DC.
- Needleman, H. L., Gunnoe, C., Leviton, A., Reed, R., Peresle, H., Moher, O., and Barrett, P. (1979). Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N. Engl. J. Med.* 300, 689-693.
- New Jersey State Department of Health, Division of Occupational and Environmental Health. (1985). Unpublished data on lead in soil.
- Roels, H. A., Buchet, J. P., Lauwerys, R. R., Bruaux, P., Claeys-Thureau, P., Lafontaine, A., and Verduyn, G. (1980). Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. *Environ. Res.* 22, 81-94.
- Schnitt, N., Phillon, J. J., Larsen, A. A., Harnadek, M., and Lynch, A. J. (1979). Surface soil as a potential source of lead exposure for young children. *CMA J.* 121, 1474-1478.
- Spittler, T. M., and Feder, W. A. (1979). A study of soil contamination and plant lead uptake in Boston urban gardens. *Commun. Soil Sci. Plant Anal.* 10, 1195-1210.
- U.S. Environmental Protection Agency, Office of Research and Development. (1983). "Air Quality Criteria for Lead," Vol. I. EPA Report No. EPA-600/8-83-028A. U.S. EPA, Research Triangle Park, NC.
- Wilson, K., and Crouch, E. (1982). "Risk/Benefit Analysis," pp. 61-63. Ballinger, Cambridge, MA.